EXHIBIT 26

RESPONSE OF DR. FRANK AND DR. WHITEHOUSE TO GRACE'S DR. D. WEILL REPORT, 4/6/09

1. 3mm.

Dr. Weill, p.2, states: "Less than 3mm would be difficult to distinguish from normal." We find that in practice 2mm is quite distinguishable. The Dr. Henry 4/6/09 Report notes that HRCT can detect pleural thickening at perhaps the 1-2mm thickness level, citing Webb et al, High Resolution CT of the Lung, 3d. Ed. 2001, 236-51.

2. Ameille et al (2004).

Weill, p.3, cites Ameille et al (2004) for a definition of DPT requiring costophrenic angle obliteration. Ameille et al (2004), p.294, proposes that obliteration of the costophrenic angle be a criterion for diagnosis of DPT. As we have noted before, neither the ATS (2004) Official Statement, nor lung disease texts add on obliteration of the costophrenic angle (blunting) to the definition of diffuse pleural thickening.

Ameille et al (2004) cites three studies showing a correlation between blunting and decreases in FVC, FEV1, and TLC. These are Schwartz et al (1990), Lilis et al (1991), and Bourbeau et al (1990). All three studies arbitrarily define DPT as requiring blunting. Then comparisons were made with pleural plaques on loss of lung function. Not surprisingly comparing loss of lung function in patients with DPT and blunting exceeds that in patients with mainly pleural plaques. This is a comparison of apples and oranges. More interesting would be to compare loss of lung function in patients with DPT and blunting, as against patients with DPT per width and extent criteria.

There was no comparison of loss of lung function for DPT with and without blunting in the three studies above. Thus they provide no medical basis for excluding patients with DPT and no blunting from the TDP medical criteria's definition of diffuse pleural thickening.

McLoud et al (1985), Fig. 10, did compare lung function loss for three types of diffuse pleural thickening, (1) pleural thickening due to "confluent

plaques," (2) pleural thickening "post effusion," and (3) pleural thickening due to "asbestosis" ("extension of pulmonary fibrosis to visceral and parietal pleura," p.12). ATS (2004) Official Statement, p.707, concurs in the three types of diffuse pleural thickening.

The "post effusion" category can represent a blunting category since 90% of the "post effusion" cases had blunting, and these cases represented 93% of the cases of blunting (52 of 56). The results at Fig. 10 show that the three causes of pleural thickening were quite consistent in lung function loss over increasing degrees of asbestosis. "Post effusion" cases of pleural thickening incurred somewhat more loss of vital capacity, but the difference surely could not justify tossing out all cases of pleural thickening due to "confluent plaques" and/or "asbestosis" from the definition of pleural thickening. A similar pattern was found on Fig. 10 for loss of diffusion capacity (DLCO) for all three forms of diffuse pleural thickening.

The Ameille et al (2004) data at Table 4 indicate that if the definition of diffuse pleural thickening is limited to patients with blunting, then patients with DPT along over 25% of the chest wall, but no blunting, end up in the "pleural plaques" group. Then the pleural plaques group is tossed out. This " pleural plaques" group, containing many true DPT cases has 35% with exertional dyspnea and 19% with chest pain.

Interestingly, Bourbeau et al (1990), p.840, made some differentiation by "width and extent" of pleural thickening and stated:

> Although not shown in the Table, as for any pleural abnormality, complaint for dyspnea with major activities was statistically significant ($p<0.05$) when related to chest wall pleural plaques taken as a continuous variable according to width and extent independently of parenchymal disease.

Table 3 shows an odds ratio of 4.5 for "chest wall" pleural thickening, as compared to 2.1 for patients with blunting of the costophrenic angle.

Lee et al (2003) was a study of Wittenoom asbestos miners with amphibole exposure. The authors developed a DPT score, based upon width and extent of pleural thickening. Presence or absence of blunting was noted, but did not enter into the scoring. The authors found that the

"pleural thickening score correlated inversely with FEV1, FVC and TLC" (p.203). Lee et al (2003), Table 2, shows slightly lower values for FVC and TLC than does the Ameille Table 4 "DPT group." So, it does appear that there may be a similar lung function loss correlation whether the variable be blunting or width and extent of pleural thickening. And, Lee et al (2003) as an amphibole study is more relevant to Libby issues than the other three studies discussed above.

McLoud et al (1985) presents the best data on types of diffuse pleural thickening. The abstract states:

> Diffuse pleural thickening was defined as a smooth, non-interrupted pleural density extending over at least one-fourth of the chest wall, with or without costophrenic angle obliteration.

This is consistent with the standard definition for diffuse pleural thickening in ATS (2004) and various text books. McLoud et al (1985) carefully examined films for 185 patients and sorted them as follows, at Table 3:

Cause of Diffuse Thickening	No.	Percentage
Benign asbestos diffusion	58	31%
Confluent plaques	47	25%
Extension of pulmonary fibrosis (asbestosis)	19	10%
Malignancy, infection or trauma	47	25%
Obesity	5	3%
Unexplained	9	5%
Total	185	

McLoud et al (1985), p.12, apparently reports only 56 of 185 cases with blunting, or 30%. Adopting medical criteria which only recognize diffuse pleural thickening with blunting could exclude 68 cases of true diffuse pleural thickening in the first three categories in the table above (58 + 47 +

19 = 124 - 56 = 68).

Ameille et al (2004), p.294, recognizes the findings of McLoud et al (1985) that diffuse pleural thickening may be due to (1) effusions, (2) confluence of plaques, or (3) extension of pulmonary fibrosis into the pleura. McLoud et al (1985) is frequently cited on this issue, and is followed in medical texts. ATS (2004) references McLoud et al (1985) and the three types of DPT at p.707.

Ameille et al (2004), p.294, next states:

> However, there is now a large consensus that DPT is the radiographic expression of thickening and fibrosis of the visual pleura, often associated with fusion of the parietal pleura, following clearance of benign asbestos-related pleural effusion. [Hillerdahl et al., 1990; Schwartz, 1991; Solomon, 1991; Miller et al., 1993; Rudd 1996; Consensus Report, 1997; Gevenois et al., 1998; Chailleux and Letourneux, 1999; Peacock et al., 2000].

Ameille et al (2004) imply that DPT always follows pleural effusion and should be defined as such. (Indeed DPT must always be due to effusion to support the author's conclusion.)

In fact, McLoud et al (1985), p.16, states:

> We have reported previously 35 cases of benign effusion among the same survey group of 1,135 asbestos exposed workers studied in this report, a prevalence of 3.1% [8]. Residual diffuse thickening, usually with a blunted angle was noted in half of those cases (54.3%).

We did not find that any of the articles cited by Ameille above states that DPT only follows "clearance of benign asbestos-related pleural effusion." (We note that Solomon (1991) was not available, and two other articles were in French). The Ameille unsupported observation is certainly contrary to the detailed large study done by McLoud et al (1985) which documents in detail that only a minority of DPT cases result from pleural

effusion. Ameille et al could have analyzed their own data for evidence of effusions.

Ameille et al (2004), p.294, next states:

> Several studies have confirmed that DPT is preceded by a benign asbestos pleural effusion [Martensson et al., 1987; Miller and Miller, 1993].

In Miller and Miller (1993), six cases were considered. In five cases DPT was preceded by pleural effusion. One may not generalize in a sample of six cases in any event. In Martensson et al (1987), there is no mention of DPT following effusion to any large degree. Apparently only seven of 71 patients with effusions had DPT.

Ameille et al (2004), p.294, concludes:

> Obliteration of the costophrenic angle represents the sequella of pleural effusion. It therefore appears logical to propose this criterion for the diagnosis of DPT.

There is no evidence in the studies cited that DPT always results from pleural effusion. Likewise there is no evidence in the studies cited that pleural effusions always cause blunting. In McLoud et al (1985), 90% of effusions led to blunting. McLoud cites Epler et al (1985) where 54% of asbestos pleural effusions led to blunting. Blunting often accompanies DPT. However, per McLoud et al (1985), a requirement of blunting before DPT is diagnosed would exclude a majority of cases of true DPT, which do not happen to have associated blunting of the costophrenic angle, but have DPT along a significant portion of the chest wall.

Ameille et al (2004) was not a well designed study. The authors had an opportunity to use their data to determine how well chest x-rays determine true DPT and how well the suggested definitions determine true DPT. Ameille et al (2004), p.290, states that "HRCT (high resolution CT scan) was considered to be the gold standard for the diagnosis of pleural thickening and pulmonary fibrosis." The authors had 287 subjects with "pleural thickening" on CT scans. How many of the 287 had only pleural

plaques? How many had pleural thickening on CT with or without blunting (DPT per ATS (2004))? How many of these had DPT per definition one (blunting), or definition two (extent > 25% and 5mm thickness)? We are not told. It would have been best to present the data on the 287 CTs in detail so that one could examine the distribution of data.

Probably a majority of subjects on HRCT had pleural plaques only and no symptoms. Lilis et al (1992), p.50, notes that pleural plaques represent "in most exposed populations, about 80% of all pleural fibrosis." It would have been enlightening to be able to exclude the plaques only cases, and examine how well the chest x-ray definitions of DPT operated on cases confirmed by HRCT as DPT.

In the last two paragraphs of the article, the authors pick definition one (blunting) as a "much more reliable sign" of DPT in the visceral pleura than definition two (width and extent). The authors announce without presenting data that the dimensional criteria (width and extent) "cannot reliably extinguish between fibrosis of visceral and parietal pleura." If this is the key distinction, then there must be some data to support it. In practice, when reading chest x-rays and CT scans one has the same difficulty discerning the visceral pleura from the parietal pleura regardless of the definition of DPT.

Ameille et al (2004), pp.294-295, also announce that dimensional criteria (width and extent) "cannot reliably distinguish . . . between pleural fibrosis and subpleural fat pads." Again, no data is presented in support of the assertion. Again, in practice when reading chest x-rays and CT scans one has the same difficulties discerning the difference between pleural fibrosis and subpleural fat pads, regardless of the definition of DPT. Any difficulty in discerning pleural fibrosis from fat pads does not argue for a definition of a DPT that requires blunting. Blunting has nothing to do with it. The authors may have had the data to answer questions of discernment of true DPT per HRCT reading, versus subpleural fat pads. Discernment is easier on HRCT. But, the authors did not present the data.

Effusions occur without resulting in blunting. In Epler et al (1985) blunting is seen in the absence of any evidence of effusion in 46% of cases. DPT is often seen without blunting, McLoud et al (1985). DPT is usually seen without evidence of effusion. Where DPT is seen over 25% of the chest wall and over 3mm in thickness, with or without blunting, it should be

called diffuse pleural thickening. It should not be called a plaque, where it is diffuse, non-circumscribed, and blended on the edges. Where there is no blunting, the ILO system calls diffuse pleural thickening a "pleural plaque." This is a false result.

Weill, p.4, states: "A similar reduction in lung function has been found when CAO (costophrenic angle obliteration) and DPT (diffuse pleural thickening) are considered separately," citing Bourbeau et al (1990) and Lilis et al (1992).

It does not appear that in Bourbeau et al (1990) blunting and diffuse pleural thickening (DPT) were considered separately. The article at p.838 explains:

> Because a previous study from our laboratory using the same readers suggested that confluent pleural plaques and diffuse thickening could not be reliably distinguished using the criteria stated in the ILO 1980 instructions (25), our readers were instructed to consider diffuse thickening to be present only when there was blunting of the costophrenic angle.

Defining DPT to require blunting prevents the two from being considered separately. It appears from the study, p.839, that "52.5% had pleural plaques only and 5.5% diffuse pleural thickening." This adds to nearly the 58.2% in Table 1 for "any pleural thickening." The authors state "the low prevalence of diffuse pleural change when compared with a recent study of a similar work force (12) resulted from our insistence on the presence of costophrenic angle obliteration for its reading." *Id.* This recognizes that there is considerable diffuse pleural thickening not covered by a definition that requires blunting.

Since DPT with blunting was the only definition considered, no comparison can be made between lung function loss due to DPT with and without blunting.

Nor does it appear that in Lilis et al (1992) blunting (CAO) and DPT were considered separately. Page 50 states: "Diffuse pleural fibrosis, defined as that which includes blunting the ipsilateral costophrenic angle." Since DPT is defined as that which includes blunting, the two cannot be

considered separately. Lilis et al (1992), p.50, refers to "circumscribed pleural fibrosis, which represents, in most exposed populations, about 80% of all pleural fibrosis." One must exclude the cases of pleural plaques only, then compare cases of DPT with and without blunting to determine whether cases without blunting have significant decrements of lung function. In Libby, the CARD mortality study shows that they do.

3. Spirometry.

Weill, pp.5-6, lists certain test requirements and concludes: "Whitehouse did not provide sufficient information to assess any of these criteria." This statement demonstrates a lack of familiarity with modern computerized lung function testing. Weill assumes that all data from PFT tests are placed into the patient's chart. This is not how computerized PFTs are done. If technical aspects of a PFT are not acceptable, or if repeatability requirements are not met, the computer will not print out a result. Only the best test is printed out and placed in the patient's chart.

Results are stored in the hard drive by patient/visit/test. Data on the sequence of acceptability software requirements remains in the hard drive. An investigator cannot assume, as Weill assumed, that if the internal software routines are not printed out, that they were not done. It is quite the opposite. If the software requirements for acceptability and repeatability are not met, there are no results to print out.

As the respiratory therapist proceeds through the lung function test, each one of the acceptability requirements must be met. If so a "acceptability flag" or green light appears on the computer. If the test is not acceptable, the respiratory therapist must start anew. For various tests, graphs will appear on the screen and the respiratory therapist can know how the test is proceeding, and where an unacceptable episode may have occurred.

Dr. Whitehouse has been using computerized PFT equipment since the 1980s. Whitehouse (2004) states that tests were done a Medigraphics Model 1085. There are regular consultations with experts at Medigraphics on aspects of computerized pulmonary functioning testing. It is standard practice in the Northwest to use computerized PFT equipment. It is standard practice to print out and include in the patient's chart only the best effort.

The CARD Clinic exceeds the minimum requirement of three tests. Over the last 15 years Dr. Whitehouse has reviewed hundreds of charts from other pulmonologists. It is rare indeed to see more than one tracing in a chart.

The PFTs done at CARD in Libby meet all ATS requirements for pulmonary function testing. The computer software is designed to ensure compliance. The respiratory therapists are trained to ensure compliance.

The Expert Report of Dr. Alan C. Whitehouse 12/29/08, ¶ 71, describes a comparison of CARD PFTs with those from pulmonary labs at hospitals in Kalispell and Missoula, Montana. There were 22 cases where patients had independent PFTs within six months of a CARD PFT. Differences in forced vital capacity (FVC), total lung capacity (TLC), and diffusion capacity (DLCO) were compared. The CARD results were 3.68% higher than the other pulmonary labs, which is within the margin of error for testing. Also the fact that the CARD respiratory therapists obtained higher results (closer to normal) indicates that the respiratory therapists at CARD are of higher competence.

Weill, pp.5-6, refers to specific aspects of spirometry testing, such a three traces, plateau, six seconds exhalation time, spirometry graphs and FEV1 and FVC within 0.15 litters of each other. The computerized equipment monitors all these items, as is generally explained at ATS/ERS (2005) "Standardization of Spirometry," Eur Respir J 2005, 26:153-161. As described above, detailed information on testing is stored in the computer hard drive. It is not placed in the patient's chart.

4. DLCO testing.

Weill, p.8, lists certain testing requirements and concludes "there is no information in any of Dr. Whitehouse's reports that allows us to determine if he complied with any of these criteria." The same comments on spirometry above apply to DLCO testing. Inspired volume and washout volume are monitored on the computer. If the requirements are not met, the test is unacceptable. The CARD Clinic generally obtains 3-4 acceptable DLCO trials. This is in excess of the minimum of two. Detailed information on testing is stored in the computer hard drive. It is not placed in the patient's chart.

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DATED this 14 day of May, 2009.

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